In the Specification:

Amend the paragraph beginning on page 20, line 21 as follows.

Fig. 4 shows the structure of the *C. elegans* MOD-1 amino acid sequence.

Amend the paragraph beginning at page 23, line 15 as follows.

The *mod-1* mutants, as described in the previous section, were further characterized using this technique. Animals carrying the n3034 mutation (Fig. 7 and Fig. 8) exhibited a dominant phenotype of insensitivity to exogenous serotonin in liquid locomotion assays. Animals carrying the ok103 mutation (Fig. 5 and Fig. 6) exhibited a recessive phenotype of insensitivity to exogenous serotonin in liquid locomotion assays.

a1

Amend the paragraph beginning at page 23, line 23 as follows.

Both the wild type (Fig. 2), and mutant *mod-1* cDNA have been obtained. The dominant serotonin resistance phenotype of animals carrying the *mod-1(n3034)* allele was used to genetically map *mod-1(n3034)* to a 0.7 map-unit interval on chromosome V. Deficiency analysis showed that the dominant serotonin resistance phenotype is not due to a haploinsufficiency of the *mod-1* locus. The recessive nature of the serotonin resistance phenotype at early time points was exploited to perform standard transformation rescue experiments, and subsequently, the gene was cloned (Fig. 1).

a2

Amend the paragraph beginning at page 24, line 9 as follows.

The protein encoded by the *mod-1* open reading frame responsible for the rescue is structurally similar to ligand-gated ion channels that belong to the nicotinic acetylcholine receptor (nAchR) family (Fig. 3 and Fig. 4). The nAchR family members are all pentameric channels with large N-terminal extracellular ligand-binding domains, four highly conserved transmembrane domains (M1-M4), and relatively divergent cytoplasmic domains between M3 and M4. nAchR family members include channels gated by acetylcholine, glycine, GABA, avermectin, and serotonin. Within the members of the nAchR family, structure-function analysis has been performed primarily on the acetylcholine receptor, but many structural and functional parallels have been seen with the other family members as well. In addition, chimeric channel studies show that there is a great deal of conservation at the functional level, even across the different ligand-gated members of the family. The M2 domains of the various subunits are predicted to line the pore of the channels. Site-directed mutagenesis studies of residues within this domain have demonstrated that ion specificity and modulation of the magnitude and frequency of current flux are determined, at least in part, by the residues that line the pore and those that are immediately adjacent to the pore on both the extracellular and cytoplasmic sides. Based on primary sequence analysis, MOD-1 appears to be equally divergent from all cloned nAchR family members.

Amend the paragraph beginning at page 25, line 5 as follows.

MOD-1 was heterologously expressed in *Xenopus* oocytes, injected with 50 nl of *C. elegans* RNA, or MOD-1 was expressed in HEK cells transiently transfected by calcium phosphate precipitation. Forty-eight to 72 hours later, the cells or oocytes were screened under a voltage clamp (Figs. 9A-9C). Application of 100 nM serotonin elicited large inward currents at a holding potential of -70 mV. Uninjected oocytes and nontransfected cells had no response to 10 μM serotonin. Application of 1 mM of other agonists of ligand-gated ion channels, such as acetylcholine, GABA, or glycine elicited little or no response from the MOD-1 channel.

Amend the paragraph beginning at page 26, line 2 as follows.

Ion selectivity was determined by measuring changes in the reversal potential (voltage at which the serotonin response changes from an inward, negative, to an outward, positive, current) in response to varying the ionic composition of the bath solution. The reversal potential was insensitive to changes in cations (Na^+ or K^+), but shifted by approximately 50 mV for each 10-fold change in extracellular chloride concentration (Fig. 10).

In the Claims:

Cancel claim 16 without prejudice.